

Response to Letter to the Editor “Adverse outcomes in anticoagulated patients undergoing percutaneous left atrial appendage ligation” by Anetta Undas

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Adv Interv Cardiol 2020; 16, 2 (60): 233–234

DOI: <https://doi.org/10.5114/aic.2020.96076>

We thank Prof. Undas for her interest in our recent article and comments on left atrial appendage occlusion [1]. We agree that our study indirectly points to potential benefits of non-vitamin K oral anticoagulants' (NOAC) use in selected atrial fibrillation (AF) patients following left atrial appendage occlusion (LAAO) in the setting of high thromboembolic and bleeding risk, but a dedicated study is urgently needed to support this concept. Several points need to be discussed.

We presented the real-life observational data on different anticoagulation strategies in atrial fibrillation (AF) patients after LAAO using the LARIAT system between 2009 and 2015 [1]. During this period, the guidelines on oral anticoagulation in AF shifted towards the novel drugs. NOACs were approved in adults with non-valvular AF at the end of 2011, and 18% of patients studied by us received drugs from this group at the end of the study (2015). We observed a progressive increase in NOAC use starting from 2012–2013, which is in concordance with other Polish data [2]. The fraction of patients receiving NOACs is rather small, which may be a result of the relative unfamiliarity of LAAO procedure concepts, and/or reluctance to use drugs new to the market.

Unfortunately, detailed data on the quality of anticoagulation were unavailable in the current report, but poor quality of anticoagulant therapy is a well-known problem in AF patients. In Poland, the time within the therapeutic range (TTR) in primary care patients on vitamin K antagonist (VKA) therapy is around 55% [3], and some of the observed adverse events may, arguably, be caused by poor VKA anticoagulation management. Two hemorrhagic strokes were observed in non-anticoagulated patients and were probably caused by uncontrolled hypertension. The first of those patients, with a CHA₂DS₂-VASc score

of 6 points and a HAS-BLED score of 3 points, was managed with aspirin. In the second patient, with a CHA₂DS₂-VASc score of 2 points and a HAS-BLED score of 2 points, no anticoagulation or antiplatelet drugs were given. In the OAC group a hemorrhagic stroke occurred in 1 patient on VKA, with a CHA₂DS₂-VASc score of 2 points and a HAS-BLED score of 3 points, who had INR > 3.5 (after this event, anticoagulation was discontinued completely). There was also one case of gastrointestinal bleeding in a NOAC-treated patient with a CHA₂DS₂-VASc score of 6 points and a HAS-BLED score of 5 points (the dose of dabigatran was reduced after the event). In terms of thromboembolic events (two strokes and one peripheral embolism), all occurred during VKA therapy, and anticoagulation therapy was switched to NOACs in all cases.

Although older age is listed as a risk factor for both bleeding and thromboembolism, we did not find a relationship with the incidence of adverse events, which is in line with our previous study [4]. It would be of interest to conduct a randomized trial comparing LAAO with no subsequent anticoagulation versus different anticoagulation strategies. It cannot be excluded that in individuals with high both thromboembolic and bleeding risk, LAAO with subsequent reduced-dose NOAC may be an optimal treatment strategy (in the setting of no absolute contraindications).

Conflict of interest

This study is the result of the research grant No. UMO-2014/13/D/NZ5/01351 funded by the National Science Centre. Lakkireddy D is the Co-chair of the AMAZE trial steering committee and has received an institutional research grant. Bartus K is the recipient of research grant.

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Received: 20.05.2020, accepted: 21.05.2020.

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